AMENDMENTS TO THE SPECIFICATION

The paragraph beginning on line 4 of page 25 has been amended as follows:

FcαRIa is known to be differentially glycosylated in different cell types (33,34) and it has been shown that an altered glycosylation pattern of receptor in certain diseases such as IgA nephropathy. Work with FcγRIIIa expressed on NK cells and on monocytes-macrophages provides a framework which suggests that these glycosylation differences in FcαRIa are important. FcγRIIIa expressed on NK cells and on monocyte-macrophages is also differentially glycosylated despite identical protein cores ~~(Table 3)~~ and these different glycoforms have different affinities for ligand ~~(Figure 15)~~. The macrophage form has a lower apparent Mr, is lower in sialic acid content, and has a lower affinity for IgG (113). The broad range of apparent Mr which is greater than can be explained by known splice isoforms of FcαRI is consistent with disease-associated changes in glycosylation altering receptor function.

The paragraph beginning on line 16 of page 25 has been amended as follows:

Several lines of evidence suggest that Fc receptor polymorphisms are important in the development of disease phenotype in chronic inflammatory diseases. The present invention shows that alleles for both FcγRIIa and FcγRIIIa are altered in their representation in SLE with low binding alleles associated with the SLE phenotype and high binding alleles (176V) protecting against glomerulonephritis ~~(Table 2)~~ (4,114). It is also shown that alleles of FcγRIIIB predict the severity of glomerulonephritis in ANCA-positive vasculitis such as Wegener's Granulomatosis (Figure 8). The data presented herein shows that the novel Ser→Gly in the CK1 site of the FcαRI CY domain is markedly enriched in chronic inflammatory disease (SLE) ~~(Table 3)~~.

The paragraph beginning on line 3 of page 32 has been amended as follows:

Identification of both pre- and post-translational variations and modifications of FcαRI and their impact on FcαRI function. FcαRI has several splice variants and some investigators have hypothesized that these variants may have different functional capacities (29,30,32,112,113). No systematic evaluation of these splice variants in disease states has been previously undertaken to establish their potential relevance. Similarly, FcαRI is ~~know~~ known to be differentially glycosylated in different cell types and in different disease states (128,132). Using the glycoforms of FcγRIIIa as a model (113), the present invention shows that these alterations of glycosylation of FcαRI have different functional properties.

The paragraph beginning on line 8 of page 38 has been amended as follows:

Example 12 - Determination of cytokine promoter alleles: While some microsatellites associated with various cytokine genes have been identified, a systematic search of the 5' promoter region reference sequences for novel SNPs is not available (4,45-51,113). However, SNPs occur with reasonable frequency (approximately 1 per 500-1000 bp) and ~~that~~ at least some of these SNPs are of biological significance. Identifying such SNPs and developing both microsatellite and SNP assays for characterization of a clinical population use the techniques of Examples 1-5, as well as other techniques conventional to the art. Examples of cytokine genes are given: